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10/555,865	08/25/2006	Manuel Sarasa Barrio	105090.61194US	6865

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EXAMINER	
BALLARD, KIMBERLY	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/555,865	SARASA BARRIO, MANUEL	
	Examiner	Art Unit	
	Kimberly Ballard	1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *04 September 2009*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-19* is/are pending in the application.

4a) Of the above claim(s) *5 and 8-19* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-4,6 and 7* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☒ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *11/07/05; 05/11/06; 09/11/07*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Election/Restrictions

1. Applicant's election with traverse of Group II, claims 1-4 and 6-7, drawn to the use of a peptide of SEQ ID NO: 2 or SEQ ID NO: 3 conjugated to a protein that acts as an immunogen for the production of antibodies, in the reply filed on September 4, 2009 is acknowledged. The traversal is on the ground(s) that although the different groups are patentably distinct, all the claims require the use of the same set of short peptide sequences for the production of antibodies, either for active immunization or for passive immunization. Therefore, Applicant asserts, the searches of the claims would significantly overlap and would not present an undue search burden to examine the claims of groups II and V (drawn to the use of an antibody that specifically recognizes the beta amyloid peptide and is obtained by immunization with the peptide of SEQ ID NO: 2 or SEQ ID NO: 3) in the same application. This is not found persuasive because this application is a 371 national stage application, and therefore is subject to Unity of Invention consideration under PCT Rule 13.1 and 13.2 (see MPEP § 1800) and not U.S. restriction practice such as for applications filed under 35 U.S.C. 111. Therefore, whether or not an undue burden for examination exists is not considered relevant to the instant situation because search burden is not a factor used to determine unity of invention or lack thereof. The basis for the lack of unity in the instant application was set forth in the restriction requirement mailed May 19, 2009. Specifically, because the technical feature linking Groups I-VI was disclosed in the prior art, it does not constitute a special technical feature as defined by PCT Rule 13.2, as it does not define a

contribution over the prior art. Therefore, the different groups are said to lack unity of invention.

The requirement is still deemed proper and is therefore made FINAL.

2. Claims 5 and 8-15 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to nonelected inventions, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on September 4, 2009.

3. New claims 16-19 are directed to vaccines, which do not correspond to any of the original groups set forth in the restriction requirement of July 10, 2009.

Newly submitted claims 16-19 are directed to an invention that fails to share a special technical feature with the elected invention for the following reasons: as set forth in the restriction requirement mailed July 10, 2009, the technical feature linking Groups I-VI, and of new claims 16-19, appears to be that they all relate to a peptide conjugate comprising various peptide fragments of amyloid-β conjugated to a protein carrier. However, WO 99/27944 by ATHENA NEUROSCIENCES (published June 10, 1999; listed on IDS) teaches the use of compositions comprising Aβ or an active fragment linked to a conjugate molecule that promotes delivery of Aβ to the bloodstream of a patient and/or promotes an immune response against Aβ (see paragraph spanning pp. 4-5). The WO document also discloses the production and use of an anti-Aβ antibody such as for the manufacture of a medicament for prevention or treatment of Alzheimer's

disease (see p. 5, lines 17-20). In particular, the Aβ peptides for use in the disclosure include fragments which comprise the instantly recited sequences of SEQ ID NO: 1-4. Non-limiting examples of Aβ peptides for use as conjugated immunogens include Aβ1-12, 13-28, 17-28, 25-35, 35-40, and 35-42. The reference also generically discloses active fragment of Aβ that contain an epitope, wherein the immunogenic fragments of Aβ typically have a sequence of at least 3, 5, 6, 10 or 20 contiguous amino acids from the natural peptide (see page 15, lines 1-8). Thus, the technical feature linking the inventions of Groups I-VI and of new claims 16-19 does not constitute a special technical feature as defined by PCT Rule 13.2, as it does not define a contribution over the prior art.

Applicant has elected Group I, directed to a method of treatment using a peptide conjugate. Accordingly, claims 16-19 are withdrawn from consideration as being directed to a non-elected invention. See 37 CFR 1.142(b) and MPEP § 821.03.

4. Claims **1-4** and **6-7**, drawn to the extent of the peptides of SEQ ID NO: 2 and SEQ ID NO: 3 for use in the claimed method, are under examination in the current office action.

Information Disclosure Statement

5. The information disclosure statements (IDSs) submitted on November 7, 2005, May 11, 2006, and September 11, 2007 have been considered and are of record. It is

noted that the 05/11/2006 IDS contains duplicate listings for references listed on the earlier filed (11/07/2005) IDS. Duplicate references have been lined through.

Claim Objections

6. Claims 4, 6 and 7 are objected to because of the following informalities:

Claim 4 recites “The method according to claim 1, of claim 1...", which is redundant. Appropriate correction is required.

Regarding claims 4, 6 and 7, each of the claims appear to recite typographical errors, such as at line 9 of claim 4, line 7 of claim 6, and line 7 of claim 7. Each claim recites "addition of amino acid *resides*" (emphasis added), which should be “residues”. Also, claims 6 and 7 recite “the residues of amino acids” at lines 4-5 of each of the claims, which is redundant. Appropriate correction is required.

Claim Rejections - 35 USC § 112, first paragraph

7. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 1-4, 6 and 7 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for:

> A method for the treatment of a disease characterized by the abnormal accumulation of amyloid deposits in the brain of a patient, comprising administering to a patient in need thereof an effective amount of an amyloid β

peptide conjugated to a protein immunogen for the production of antibodies that specifically recognize any of the predominant variants of Aβ40 or Aβ42 peptide, wherein the Aβ peptide is SEQ ID NO: 2, SEQ ID NO: 3, a peptide comprising at least 5 contiguous amino acid residues of SEQ ID NO: 2 or 3, or a peptide resulting from lengthening by addition of linker amino acid residues appropriate for conjugating the protein to the peptide of SEQ ID NO: 2 or SEQ ID NO: 3,

does not reasonably provide enablement for a method for the prevention of a disease characterized by amyloid deposits in the brain as broadly claimed comprising the administration of any Aβ peptide fragment resulting from shortening by elimination of amino acids of the N-terminal and/or C-terminal ends of SEQ ID NO: 2 or SEQ ID NO: 3. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The factors to be considered in determining whether a disclosure would require undue experimentation include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art and, (8) the breadth of the claims. *In re Wands*, 8 USPQ2d, 1400 (CAFC 1988).

The claims are drawn to a method for the prevention and/or treatment of a disease characterized by the accumulation of amyloid deposits in the brain of a patient, comprising administering to a patient in need thereof an effective amount of a peptide

conjugated to a protein immunogen for the production of antibodies that specifically recognize any of the predominant variants of a beta amyloid Aβ40 or Aβ42 peptide. Dependent claims recite that the disease is Alzheimer's disease (AD), the protein is keyhole limpet hemocyanin protein (KLH), and the peptide is a peptide of SEQ ID NO: 2, SEQ ID NO: 3, or peptides resulting from shortening or lengthening the peptide sequence of SEQ ID NO: 2 or SEQ ID NO: 3. It is noted that SEQ ID NO: 2 is the peptide Aβ33-40, and SEQ ID NO: 3 is the peptide Aβ33-42. Thus, the claims broadly encompass the prevention of amyloidogenic disease, such as the neurodegenerative disease of AD, with a peptide conjugate comprising a Aβ peptide fragment which, through shortening of the peptide sequence, may no longer comprise an antigenic epitope.

The nature of the invention is the disclosure that vaccination of rabbits with Aβ peptide-KLH conjugates results in the production of anti-Aβ antibodies. The instant specification indicates that diseases characterized by amyloid deposits include Alzheimer's disease, Icelandic hereditary syndrome, multiple myeloma, spongiform encephalitis (including Creutzfeldt-Jakob disease), and cerebral amyloid angiopathy (see p. 3). The specification does not explicitly define the terms “prevention” or “treatment”, but merely states that “in accordance with an aspect of the present invention, it is possible to prevent the progress of, reduce the symptoms of and/or reduce the deposition process of amyloid in an individual” (p. 4, lines 19-21). It is noted, however, that the instant application provides no working examples or evidence

demonstrating that administration of the claimed peptide conjugates to a subject in need in any way prevents a disease characterized by brain amyloid deposits.

The state of the art at the time of filing recognizes that immunization of transgenic mice which over-express APP (amyloid precursor protein; a mouse model of Alzheimer's disease) with Aβ peptides induces the production of anti-Aβ antibodies, and that that the antibodies produced therefrom facilitate a reduction in Aβ plaque burden in the brains of the transgenic mice (see, for example, Schenk et al. *Nature*, 1999; 400:173-177; reference AW on 11/07/2005 IDS). Transgenic PDAPP mice immunized with Aβ peptide also demonstrate reduced cognitive dysfunction, with improvements observed in spatial memory (see Janus et al. *Nature*, 2000; 408:979-982; Morgan et al. *Nature*, 2000; 408:982-985). Thus the art recognizes that immunization with Aβ peptides may be a valid treatment approach for reducing plaque burden or improving cognitive function in Alzheimer's disease patients. However, Solomon (*Expert Opin. Biol. Ther*. 2002; 2(8):907-917) notes that when the same strategy was actually applied to humans, it was met with less than satisfactory results. For example, in a human clinical trial involving immunization of mild- to moderate-stage Alzheimer's disease patients with Aβ peptide and an adjuvant, a small number of patients were reported to develop inflammation in the central nervous system, such that the trial had to be abruptly halted (see p. 913). None of the numerous animal studies in several different species performed prior to the clinical study indicated any adverse autoimmune reactions (such as encephalitis), so this result in humans was largely unexpected on the

part of the researchers. Thus, the relevant art recognizes unpredictability in the extrapolation of laboratory observations to clinical efficacy.

Moreover, both at the time of filing and now, effective therapy for the treatment and prevention of neurodegenerative diseases such as Alzheimer's disease, Creutzfeldt-Jakob disease, and cerebral amyloid angiopathy has eluded researchers. For instance, Vickers (*Drugs Aging*. 2002; 19(7):487-494) notes that there is no effective treatment currently available to reverse, slow down, or prevent the course of Alzheimer's disease or most other neurodegenerative brain diseases and conditions. And Mitchell et al. (*Curr Drug Targets*. 2007; 8(7):832-838) notes that while there is considerable potential to reduce the risk of hypertensive intracerebral hemorrhages (ICHs) by treatment with antihypertensive medication, as yet no effective preventative treatment for cerebral amyloid angiopathy related ICH has emerged (see abstract).

While the skill level in the art is high, the level of predictability is low. “Prevention” is generally understood in the art to encompass protection from disease or injury. Thus, given the high level of required effect, a high level of evidence showing prevention is also required. The limited examples in the instant specification directed to the induction of an anti-Aβ immune response in healthy rabbits and the examples in transgenic PDAPP mice in the prior art, however, do not provide substantial guidance commensurate in scope with the broadly claimed prevention of any disease characterized by brain amyloid deposits. Moreover, the broadest reasonable interpretation of the claims regarding a disease characterized by the accumulation of amyloid deposits in the brain of a patient would include the disease of Down’s

syndrome, which is a genetic disease caused by the nondisjunction of chromosome 21 (trisomy 21). At present, there is no known medical treatment to correct this genetic abnormality, and it is unreasonable to believe that administration of the claimed peptide conjugate could in any way prevent this disease, even if it was to be administered *in utero*.

Furthermore, the claims are directed to methods of using fragments of Aβ peptides which may be too short to effectively induce an immunogenic antibody response. Claims 4, 6 and 7 recite that the Aβ peptide may be one resulting from shortening by elimination of amino acids of the N-terminal and/or C-terminal ends of SEQ ID NO: 2 or SEQ ID NO: 3 (e.g., Aβ33-40 and Aβ33-42, respectively). While shortening the C-terminal end of Aβ33-42 to give Aβ33-40 may obviously be acceptable, the claims are not limited to a maximum number of residues that may be eliminated, and thus read on peptide fragments having as few as one or two amino acid residues. As the peptide epitope length decreases below an optimal length – generally considered in the art to be a hexamer (see Hopp et al. *Proc Natl Acad Sci*. 1981; 78(6):3824-3828) – the antibody response generated upon administration of the peptides becomes less specific for the parent antigen, and therefore the predictability of therapeutic efficacy would similarly be reduced. Yednock et al. (US 2006/0188512 A1) notes, for example, that it is preferable to administer Aβ peptides fragments which lack a T-cell epitope because these epitopes may cause undesired inflammatory responses in a subset of patients (see [0033]). Generally, T-cell epitopes are greater than 10 contiguous amino acids. Therefore, Yednock teaches, preferred fragments of Aβ are

noted to be 5-10 amino acids in length, i.e., sufficient in length to generate an antibody response without generating a T-cell response (see [0033]). Therefore, the art recognizes the unpredictability of antigenic peptides of less than 5 amino acids in length for generating a specific immune response, particularly for therapeutic purposes.

The scope of the claims must bear a reasonable correlation with the scope of enablement (*In re Fisher*, 166 USPQ 19 24 (CCPA 1970)). Without such guidance, the changes which can be made and still maintain activity/utility is unpredictable and the experimentation left to those skilled in the art is unnecessarily, and improperly, extensive and undue. *See Ex parte Forman*, 230 USPQ 546 (Bd. Pat. App. & Int. 1986). Therefore, even if the claims were enabled for a method of preventing an Aβ-related disease, they would still not be enabled for the full scope of the claims encompassing the use of non-antigenic and/or non-specific fragments of Aβ peptides.

The test of enablement is not whether any experimentation is necessary, but whether, if experimentation is necessary, it is undue. Due to the large quantity of experimentation necessary to establish a preventative effect using the claimed Ab peptides, the breadth of the claims which encompasses the prevention or treatment of any disease characterized by deposition of Aβ in the brain, the state of the prior art which establishes the unpredictability of laboratory studies and the use of peptide fragments fewer than 5 amino acids in length, the lack of guidance or evidence in the specification or in the art indicating a preventative effect of the claimed peptides in a subject, and the complex nature of the invention, undue experimentation would be required of the skilled artisan to practice the claimed invention.

Claim Rejections - 35 USC § 102

9. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.
>
> (e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

10. Claims 1-4, 6 and 7 are rejected under 35 U.S.C. 102(b) as being anticipated by WO 00/72880 A2 by Schenk et al. (published December 7, 2000; reference AM on IDS filed 11/07/2005).

Schenk discloses methods of treating a disease associated with amyloid deposits of Aβ in the brain of a patient, such as Alzheimer's disease (see p. 4, lines 5-7), which addresses claim 2. The method entails administering an effective amount of fragments of Aβ to elicit an immunogenic response against certain epitopes within Aβ (see p. 4, lines 8-9), which includes both Aβ40 and Aβ42 (see p. 10, lines 9-10), thus addressing recited limitations of claim 1.

Regarding claim 3, Schenk teaches that the Aβ peptide immunogen may be coupled to a carrier protein to help elicit an immune response (see p. 28, lines 14-17). One such suitable carrier protein disclosed by Schenk is keyhole limpet hemocyanin (KLH) (see p. 28, lines 17-18).

Regarding claims 4, 6 and 7, Schenk discloses that the immunogenic fragments of Aβ have a sequence no more than 10, 9, 8, 7 or 5 contiguous residues in length (see p. 14, lines 31-32), and can include such Aβ peptide fragments as Aβ35-40, Aβ35-42 (p. 15, line 7), and Aβ33-42 (p. 66, line 19), among others. Thus, Schenk explicitly teaches the peptide of instant SEQ ID NO: 3 (Aβ33-42), and also teaches fragments having no more than 8 amino acid residues, which would include the peptide of instant SEQ ID NO: 2 (i.e., Aβ33-40), which is a peptide resulting from shortening by elimination of 2 amino acids of the C-terminal end of SEQ ID NO: 3.

Accordingly, the teachings of Schenk anticipate the present invention of claims 1-4, 6 and 7.

11. Claims 1-4, 6 and 7 are rejected under 35 U.S.C. 102(e) as being anticipated by US 2006/0188512 A1 by Yednock et al. (published August 24, 2006; priority to February 1, 2003).

Yednock teaches methods for treating a disease associated with amyloid deposits using fragments from a central or C-terminal region of Aβ, wherein administration of the fragments induces a polyclonal mixture of antibodies that specifically bind to soluble Aβ to inhibit formation of amyloid deposits in the brain of a patient, thus treating the disease (see [0009]). The predominant natural forms of Aβ, also known as β-amyloid peptide, include Aβ39, Aβ40, Aβ41, Aβ42 and Aβ43 (see [0016]). Such would address recited limitations of claim 1 regarding the treatment method and the predominant amyloid species. Further, Yednock discloses that the

peptide immunogens can be linked a single carrier, such as a carrier protein that is a Th cell epitope, to form a conjugate (see [0045]), which meets recited limitations of claim 1 regarding a peptide (i.e., Aβ peptide) conjugated to a protein immunogen (i.e., a carrier protein).

Regarding claim 2, Yednock teaches methods for the treatment of Alzheimer's disease (see Abstract).

Regarding claim 3, keyhole limpet hemocyanin (KLH) is disclosed as a suitable carrier protein at [0045].

And with respect to claims 4, 6 and 7, Yednock teaches that C-terminal fragments of Aβ42 of 5-10 or preferably 7-10 contiguous amino acids are considered for peptide immunogens (see [0034]). Yednock discloses administering an effective regiment of a fragment of Aβ, such as Aβ33-42 or Aβ35-40, for induction of an immune response for therapy (see Yednock's claim 48 at p. 25). Hence, Yednock teaches the peptide of SEQ ID NO: 3, and a peptide resulting from shortening by elimination of residues of the N-terminal end of SEQ ID NO: 2 (i.e., Aβ35-40). Additionally, Yednock discloses immunogenic fragments of Aβ flanked by polylysine sequences (see [0040]), as well as the use of spacer residues (e.g., Gly-Gly) between the T_h epitope and the Aβ peptide immunogen (see [0050]), thus addressing a recited limitation of the claims regarding peptides resulting from lengthening by addition of amino acid residues necessary for protein conjugation.

Conclusion

12. No claims are allowed.

13. The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.

WO 00/50077 by COSTE, M et al. Published February 22, 2000.

US 2003/0073655 A1 by Chain DG. Published April 17, 2003.

Advisory Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Kimberly Ballard whose telephone number is 571-272-2150. The examiner can normally be reached on Monday-Friday 8:30 AM - 5 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Stucker can be reached on 571-272-0911. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

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